DOES DEXAMETHASONE IMPROVE THE QUALITY OF INTRAVENOUS REGIONAL ANALGESIA WHEN ADDED WITH LIGNOCAINE-A COMPARATIVE STUDY

A STUDY OF 75 CASES

DISSERTATION SUBMITTED FOR

DOCTOR OF MEDICINE

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CERTIFICATE

This is to certify that this dissertation entitled **"DOES DEXAMETHASONE IMPROVE THE QUALITY OF INTRAVENOUS REGIONAL ANALGESIA WHEN ADDED WITH LIGNOCAINE-A COMPARATIVE STUDY"** submitted by **DR.PRAVEEN.D** to the faculty of ANAESTHESIOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement in the award of degree of M.D.Degree, Branch-X (ANAESTHESIOLOGY), for the March 2010 examination is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, **Dr.PRAVEEN.D** declare that the dissertation titled **"DOES DEXAMETHASONE IMPROVE THE QUALITY OF INTRAVENOUS REGIONAL ANAESTHESIA - A RANDOMIZED CONTROLLED CLINICAL STUDY**" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D.Degree, Branch X (ANAESTHESIOLOGY) degree Examination to be held in March 2010.

Place : Madurai

Date : **Dr. PRAVEEN.D**

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CONTENTS

INTRODUCTION

Intravenous Regional Anaesthesia (IVRA) since its birth in the hands of August Bier in 1908 has become a valuable instrument in the repertoire of anaesthesia providers. This method enjoyed wide popularity for a long time. It was not long before simple and reliable techniques for brachial plexus developed, and the intravenous method declined in popularity.

It was revived in 1963 by Holmes, who used lignocaine because it appeared to give more reliable anaesthesia than procaine. With slight technical modifications IVRA, today is an ideal method of providing anaesthesia for minor surgical procedures to the extremities performed on an ambulatory basis. It has the advantages of speed of onset, rapid recovery, reliability of blockade & cost effectiveness.

Adjuvants to local anaesthetics have greatly expanded the potential applications of regional anaesthesia by providing faster onset time, inhibition of tourniquet pain, prolonged post-operative

anaesthesia and improved peri-operative analgesia apart from decreasing risk of local anaesthetic toxicity.

In this regard, DEXAMETHASONE – a parenterally administered steroidal drug by decreasing tissue prostaglandin (PG) and other inflammatory mediator synthesis, decreases peri-operative pain in combination with LA.

Cumulative effects of these agents result in greater patient satisfaction, rapid hospital discharge , cost effectiveness and minimal risks.

AIM OF THE STUDY

To prove the effectiveness of DEXAMETHASONE as an adjuvant in Intravenous Regional Anaesthesia and to know its effectiveness in relieving tourniquet pain & providing post operative analgesia in IVRA.

INTRAVENOUS REGIONAL ANAESTHESIA

Intravenous regional anaesthesia (IVRA) was first described by August Bier in 1908. He observed that when local anaesthetic was injected intravenously between two tourniquets on a limb, a rapid onset of anaesthesia occurred in the area between the tourniquets and a slower onset occurred beyond the distal tourniquet. The technique did not become popular until the 1960s when it was reintroduced by Holmes. Today, the technique is slightly modified, using either a single or a double tourniquet at one site and injecting local anaesthetic as distal as possible to the cuff. The double tourniquet is used to increase safety and to reduce tourniquet pain in the awake patient, but there is a possibility of accidental deflation of the wrong cuff, which may lead to toxic systemic levels of local anaesthetic.

IVRA is technically simple and does not require specific anatomical knowledge. Success rate is 96–100% with a low incidence of side-effects. It is a reliable, simple and safe method of providing anaesthesia for minor surgical procedures to the extremities if it is administered by experienced clinicians.

ADVANTAGES AND DISADVANTAGES OF INTRAVENOUS REGIONAL ANAESTHESIA

Advantages :

- Speed of onset and rapid recovery
- Reliability (in the absence of local infection and with adequate equipment)
- Muscle relaxation
- Technical simplicity

Disadvantages and Complications

- Poor postoperative analgesia
- Limited time of surgical anaesthesia (< 90 minutes)
- The potential of systemic local anaesthetic toxicity
- Nerve damage secondary to direct compression by the tourniquet.
- Compartment syndrome and loss of limb (very rare)

Mechanisms of action:

Local anaesthetic diffuses into the small veins surrounding the nerves and then into the vasa nervorum and capillary plexus of the nerves, leading to a core to mantle (centrifugal) conduction block in the nerves involved. Local anaesthetic then diffuses into the small nerves in the skin, blocking their conduction. The tourniquet

produces ischaemia, which contributes to the analgesic action of the local anaesthetic by blocking nerve conduction and motor endplate function. 20 minutes after tourniquet application alone there will be analgesia to pinprick without the injection of any local anaesthetic. However, the speed of onset and the density of anaesthesia are greater with injection of local anaesthetic.

Indications:

IVRA is used for surgical interventions on the hand, forearm or elbow that will not exceed 1 hour. These include manipulation of forearm fractures, excision of wrist ganglia and palmar fasciotomy. IVRA is particularly useful for tendon grafting because it enables the surgeon to observe movement and tension of the grafted tendon (after deflating the tourniquet) before closing the wound (continued anaesthesia with a wrist block). IVRA can also be used for surgery on the foot, ankle or lower leg, for example for removing plates, screws or foreign bodies. Surgery on the elbow or knee is poorly tolerated using IVRA.

Contraindications are mainly related to tourniquet use. Absolute contraindications include sickle cell disease, Raynaud's disease or scleroderma, allergy to local anaesthetics and patient refusal. Relative contraindications include severe hypertensive or peripheral vascular

disease, local infection, and skeletal muscle disorders or Paget's disease (local anaesthetic may spread to the systemic circulation via venous channels in bone).

Procedure

Before the procedure the patient should be:

- starved for 6 hours
- monitored closely (standard monitoring applied)
- placed on a tipping trolley
- adequately informed about the procedure and should have consented to it.

The equipment required for IVRA includes:

- pneumatic tourniquet (checked for leaks before the procedure) and a pressure gauge
- Esmarch bandage or Rhys-Davis exsanguinator
- local anaesthetic solution
- resuscitation equipment and drugs.

IVRA of the arm:

A 22 G cannula is placed intravenously as distal as possible in the arm to be anaesthetized. Venous access is established in the opposite arm to allow administration of fluids or drugs if necessary. The double tourniquet (two tourniquets each 6 cm wide) or a single one (14 cm wide) is applied on the arm with generous layers of

padding, ensuring that no wrinkles are formed and the tourniquet edges do not touch the skin. The arm is exsanguinated either by using the Esmarch bandage or a Rhys-Davis exsanguinator. If this is impossible, exsanguination can be achieved by elevating the arm for 2–3 minutes while compressing the axillary artery. The distal tourniquet is inflated to at least 100 mmHg higher than the patient's systolic blood pressure (250–300 mmHg). The proximal tourniquet is inflated to the same pressure. After ensuring inflation, the distal cuff is deflated.

Before injecting local anaesthetic it must be confirmed that no radial pulse is palpable. The local anaesthetic is then injected slowly. A standard volume for injection into the upper limb is 40 ml, which can be increased to 50 ml in a fit, large adult. If the injection is too rapid, the venous pressure may exceed the tourniquet pressure and the local anaesthetic solution may escape into the systemic circulation. Surgical anaesthesia is usually achieved within 15 minutes. The distal tourniquet, which overlies part of the anaesthetized arm, can then be inflated and the proximal one deflated to relieve tourniquet pain.

The cuff should not be deflated until 20 minutes after local anaesthetic injection because systemic toxic doses of local

anaesthetic may occur. After 20 minutes, 30% of the injected drug is fixed within the tissues and is unavailable for immediate release into the systemic circulation. Cuff deflation should be performed in cycles with deflation/inflation times of less than 10 seconds until the patient no longer exhibits signs of systemic toxicity (e.g. tingling of the lips, tinnitus, drowsiness). Severe signs of systemic toxicity include bradycardia, hypotension, ECG abnormalities, seizures and loss of consciousness. Maximum blood levels of local anaesthesia occur within 10 minutes of cuff deflation. Therefore, the patient should be monitored closely for 30 minutes following tourniquet release. With lignocaine, 2.5–3 mg/kg, and cuff deflation after 10 minutes, blood levels have been reported to be less than 2 micrograms/ml.

If severe CNS intoxication occurs, appropriate resuscitation guidelines should be followed. Emergency drugs must be readily available and 100% oxygen should be administered.

IVRA of the leg:

The basic technique is the same as for the arm but the dose and volume of local anaesthetic has to be doubled for IVRA of the leg, which is associated with an increased potential for local anaesthetic toxicity. The tourniquet pressure must be higher in the leg (350–400 mmHg), to occlude blood flow in the femoral artery. This may

increase the occurrence of tourniquet pain. Tourniquets may be applied to the thigh (two tourniquets about 9 cm wide) or one at the calf (below the head of the fibula) and one at the thigh. The latter is for safety in case of distal cuff failure and is not usually inflated.

Choice of drugs

Many local anaesthetic drugs, with or without additives, have been used for IVRA, but 0.5% prilocaine, 3–6 mg/kg, is the drug of choice because it has less systemic toxicity and is partially taken up in the lungs before reaching the systemic circulation. The usual dose is 40 ml (200 mg) without epinephrine. However, the manufacturers have ceased production of 0.5% prilocaine. 1% prilocaine remains available and is licensed for IVRA, though its stability is not guaranteed if diluted. If prilocaine is unavailable 0.5% lignocaine, 3 mg/kg, is used. If IVRA is applied to the leg a larger volume must be injected (up to 100 ml). Prilocaine can be used undiluted (maximum recommended dose is 400 mg in adults) but lignocaine is commonly diluted to lower concentrations (e.g. 0.2–0.25%).

Prilocaine can cause methaemoglobinaemia but unless doses in excess of 600 mg are used it is clinically insignificant in most patients. Although one has to be aware that in patients with anaemia or cardiac conditions even small amounts of methaemoglobin can

significantly impair the oxygen-carrying capacity of their red blood cells. Intravenous regional anaesthesia with prilocaine in these patients should be considered carefully for its benefits.

Other local anaesthetic agents have been used but do not provide superior analgesia or more rapid onset of block. Severe toxic reactions and death have been observed with bupivacaine and its use is contraindicated. In one study, 0.2% ropivacaine was intraoperatively as effective as 0.5% prilocaine but postoperative analgesia was prolonged;

Additives to local anaesthetics have not been consistently shown to have an effect during IVRA but may increase the length of postoperative analgesia, probably because of a systemic effect following tourniquet release. The reported enhancement of IVRA with pethidine, 1 mg/kg, may reflect intrinsic local anaesthetic activity of the drug.

Experiments with the addition of muscle relaxants produced marked muscle relaxation but did not augment analgesia.

Ketamine alone appears to provide good sensory analgesia but some patients lost consciousness and exhibited the typical features of ketamine anaesthesia after tourniquet release.

Many other drugs have been studied, but only the addition of clonidine, 150 micrograms, an α 2-agonist, or the non-steroidal anti-inflammatory drugs ketorolac, 20 mg, or tenoxicam, 20 mg, to the local anaesthetic solution appeared to be effective in prolonging postoperative analgesia and relieving tourniquet pain. Guanethidine and calcium-channel blockers have been evaluated in the context of chronic pain management only .

TOURNIQUET

Intravenous regional anaesthesia is a method of producing analgesia of the distal part of a limb by intravenous injection, while circulation to the limb is occluded.

Occlusion of the limb was done previously by winding an Esmarch bandage proximally up the arm. Now occlusion of the limb was achieved by pneumatic tourniquet. Unfortunately, the tourniquet is not physiologic and is associated with number of disadvantages.

SITE OF APPLICAION:

The upper arm and thigh have sufficient muscle built to distribute the cuff pressure evenly and are recommended sites.

CUFF WIDTH:

The American Heart Association concluded that if a sphygmomanometer cuff has a width of 20% greater than the diameter of the upper arm or 40% of the circumference of the thigh (to a maximum of 20cm), then the pressure in the underlying central artery will be equal to that in the cuff. Modern silicone cuffs tend to be smaller than this, measuring 90mm width (bladder 70mm) for the arm and 105mm (bladder 75mm) for the leg.

The tissues immediately underlying the cuff should be protected with cotton wool. This is not necessary with correctly applied modern silicone cuff.

PRESSURE:

It was based on the unsedated patient's blood pressure measured in the ward with the adult sized cuff. The recommended cuff pressure for the upper limb is systolic BP plus 100mmHg and for lower limb twice systolic BP. This higher pressure is needed because there is often not enough room above the operating site for full sized cuff

TOURNIQUET TIME:

The recommended time for upper limb is 90minutes. Two hours should be regarded as a maximum but this will not be safe for all patients. Notify the surgeon about tourniquet time every half an hour.

CONTRAINDICATIONS:

- Sickle cell disease
- Raynaud's disease and other peripheral vascular disease
- Tumour or severe infection at the site of application
- Severe left ventricular failure

- Deep venous thrombosis- Massive total pulmonary embolism has been reported.

PHYSIOLOGIC CHANGES CAUSED BY LIMB TOURNIQUETS:

NEUROLOGIC EFFECTS:

1. Abolition of somatosensory evoked potentials and nerve conduction occurs with in 30minutes
2. Application for more than 60minutes causes tourniquet pain and hypertension
3. Application for more than 2hours may result in postoperative neuropraxia
4. Evidence of nerve injury may occur at a skin level underlying the edge of the tourniquet

MUSCLE CHANGES:

- Cellular hypoxia develops within 8minutes
- Cellular creatine level declines
- Progressive cellular acidosis occurs
- Endothelial capillary leak develops after 2hours
- Limb becomes progressively colder

SYSTEMIC EFFECTS OF TOURNIQUET INFLATION:

Arterial and pulmonary artery pressures become elevated, although this effect is usually slight to moderate if only one limb is occluded.

SYSTEMIC EFFECTS OF TOURNIQUET RELEASE:

- Transient fall in core temperature
- Transient metabolic acidosis
- Transient fall in central venous oxygen tension
- Rapid release of acid metabolites into central circulation
- Transient fall in pulmonary and systemic arterial pressures.
- Transient increase in end – tidal carbondioxide
- Increased oxygen consumption

TOURNIQUET PAIN:

Patients receiving spinal anaesthesia may develop a poorly defined aching or burning sensation in the distal extremity about one hour after tourniquet inflation. Although the mechanism and neural pathways for this severe aching and burning sensation defy precise explanation, unmyelinated, slow –conduction C fibres, which are relatively resistant to local anaesthetic blockade, probably play a critical role. Even during general anaesthesia, tourniquet pain can be revealed by a gradually increasing mean arterial blood pressure. The

tourniquet pain and its accompanying hypertension influenced by many factors including anaesthetic technique (IVRA > Epidural> Spinal> GA), intensity and level of block, choice of local anaesthetic (hyperbaric spinal with tetracaine > isobaric bupivacaine), and supplementation of the block with opioids. Cuff deflation invariably and immediately relieves the sensation of tourniquet pain and its hypertension. Systemic opioids have questionable value in relieving tourniquet pain.

PHARMACOLOGY OF LIGNOCAINE

Lignocaine is a synthetic amide-linked anaesthetic of intermediate potency and duration. In 1943 Lofgren synthesized Lignocaine in Sweden. First used by Gordh in 1948.

Lignocaine is the standard to which all other local anaesthetics are compared. It is currently the most widely used local anaesthetic. In addition, it is a popular antiarrythmic. It can be given by almost any route.

Mechanism of action :

Lignocaine prevents transmission of nerve impulses by inhibiting passage of sodium ions through ion-selective sodium channels in the nerve membranes. This slows the rate of depolarization such that the threshold potential is not reached and thus action potential is not propagated. But resting membrane potential is not altered. Lignocaine binds to the inner portion receptor (i.e Sodium channel) after entering the cell membrane.

Physiochemical properties :

Molecular weight 234

Weak base with a pka 7.6 – 7.8

Very stable, not decomposed by boiling, acids or alkalies

It is less lipid soluble than that of Bupivacaine

Pharmacokinetics :

Absorption :

It is absorbed from the site of application or injection into the blood stream. Rate of absorption depends on the blood flow to the area and use of epinephrine.

Metabolism :

Metabolised in liver by oxidative dealkylation to monoethylglycine xylidide followed by hydrolysis of this metabolite to xylidide. Metabolism is dependant on hepatic blood flow.

Monoethylglycine xylidide has 80% activity of the parent drug.

Xylidide has 10% activity of the parent drug.

75% of xylidide is excreted in the urine as 4 – hydroxyl – 2,6 – dimethylaniline.

Onset of action :

Rapid onset of action

- Topical anaesthesia 5-10 mins
- Conduction anaesthesia

  For small nerves 5-10 mins

  For large nerves 10-15 mins
- Intravenous administration 1-2 mins

Protein binding :

It is 70% bound to α 1 acid glycoprotein.

Volume of distribution :

91 litres

Distribution :

Lignocaine has a triphasic distribution

Rapid distribution phase (α) :

In this phase, the drug is distributed to highly vascular regions.

$t^{½}$ α is 1 min.

Slow disappearance phase (β) :

The drug is distributed to slowly equilibrating tissues.

$t^{½}$ β is 9.6 min.

Slow transformation and excretion phase (δ) :

$t^{½}$ is δ is 1.6 hrs

Clearance is 0.95 litres per minute

Availability :

a) 5% heavy 2 ml ampoules which contain 50 mg of lignocaine / ml with 75 mg – 100 mg of dextrose.

b) 2% lignocaine (xylocard) without preservative – 50 ml vial for intravenous use.

c) 2% lignocaine – plain – 30 ml vial –contains methyl and propyl paraben as preservative.

d) 4% lignocaine with 1 in 200000 Adrenaline – 30 ml vial.

e) 4% lignocaine viscous

f) 4% lignocaine aqueous solution

g) 10% lignocaine spray

h) 2% lignocaine Jelly

i) 2% lignocaine ointment

j) 5% lignocaine ointment

Pharmacodynamics :

Local actions :

Causes nerve blockade with loss of pain and temperature sensation, touch, motor power and vasomotor tone in the region supplied by the nerves blocked.

Systemic actions :

Result of systemic absorption from the site of administration or intravenous administration

Cardiovascular system :

It has a stabilizing effect on the cell membranes of cardiac tissue.

Lignocaine depresses myocardial automaticity by antagonizing the spontaneous phase IV depolarization and reduces the duration of effective refractory period.

Myocardial contractility and conduction velocity are depressed at higher concentrations.

These effects result from direct cardiac muscle membrane changes (ie.) cardiac sodium channel blockade.

It stabilizes the membrane of damaged and excitable cells, tending to suppress ectopic foci.

Respiratory system :

Lignocaine depresses hypoxic drive (the ventilatory response to low P_aO_2).

Apnea can result from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to the local anaesthetic agents.

Relaxes bronchial smooth muscle.

Intravenous lignocaine may be effective in blocking the reflex bronchoconstriction associated with intubation.

Vascular smooth muscle :

Produces vasodilatation

Central nervous system :

Produces a sequence of stimulation followed by depression. Produces sedation on intravenous administration.

Intravenous administration decreases cerebral blood flow and attenuates the rise in intracranial pressure that accompanies intubation.

Infusion of lignocaine is capable of reducing the MAC of volatile anaesthetics by 40%.

Musculoskeletal :

Lignocaine is myotoxic leading to lytic degeneration, edema and necrosis.

Haematological :

It decreases coagulation and enhances fibrinolysis

Indications :

1. For infiltration block, peripheral nerve blocks, epidural, spinal and topical anaesthesia & intravenous regional anaesthesia.

2. Antiarrythmic :

Lignocaine is a class IB antiarrythmic.

Ventricular tachyarrythmias

Arrythmias following acute MI during cardiac surgery

In digitalis toxicity – because it does not worsen AV – block.

3. Prevention or treatment of increases in intracranial pressure during intubation.

- antitussive effect may be the reason.

4. Reflex induced bronchospasm is also attenuated by iv administration of lignocaine

5. Suppresses noxious reflexes such as coughing & sympathetic stimulations associated with endotracheal suctioning and intubation.

6. Used as an antiepileptic agent intravenously

7. Used intravenously as an analgesic for certain chronic pain states

8. Used as a supplement to general anesthesia.

Contraindications

1. Hypersensitivity
2. Should not be used with vasoconstrictors in digits of hands, feet and penis

Stokes Adams syndrome, severe degree of heart block

Doses :

Maximum recommended dose :

a) Plain - 3 mg / kg

b) with adrenaline- 7 mg / kg

c) for reflex suppression - 1.5 mg / kg iv.

Drug interactions :

β Blockers :

Coadministration of betablockers- increases serum levels of lignocaine and its toxicity by decreasing lignocaine's metabolism.

Anticonvulsant agents :

Increases lignocaine's metabolism

Non depolarizing muscle relaxant

Blockade is potentiated by lignocaine

Opioids and α_2 adrenergic agonists :

Potentiate lignocaine's pain relief

Antiarrythmic agents

Potentiate the cardiac effects of lignocaine

Toxicity :

Mostly due to systemic absorption of locally administered lignocaine or due to accidental intravenous administration of large doses of lignocaine.

The central nervous system is mostly vulnerable.

Blood levels and symptoms :

4 µg / ml : Light headedness, tinnitus, circumoral and tongue numbness (anticonvulsant and antiarrhythmic activity)

6 µg / ml : visual disturbances

8 µg / ml : muscular twitching

10 µg / ml : convulsions

12 µg / ml : Unconsciousness

15 µg / ml : Coma

20 µg / ml : respiratory arrest

26 µg / ml : cardiovascular collapse

Treatment of toxicity :

Continuous monitoring of CVS and RS status helps to identify the toxicity earlier.

- If convulsions occur barbiturates or benzodiazepines can be given.
- Succinylcholine 1 mg / kg to paralyse the patient and aids in controlling the seizures.
- Cardiac toxicity like fibrillation can be treated by defibrillation
- Ventilatory support – 100 % oxygenation, intubation etc.,
- Maintain B.P. by rapid infusion of I.V. fluids, use of vasopressors and put the patient in Trendelenberg's position.
- Maintain fluid and electrolyte balance.

Adverse effects :

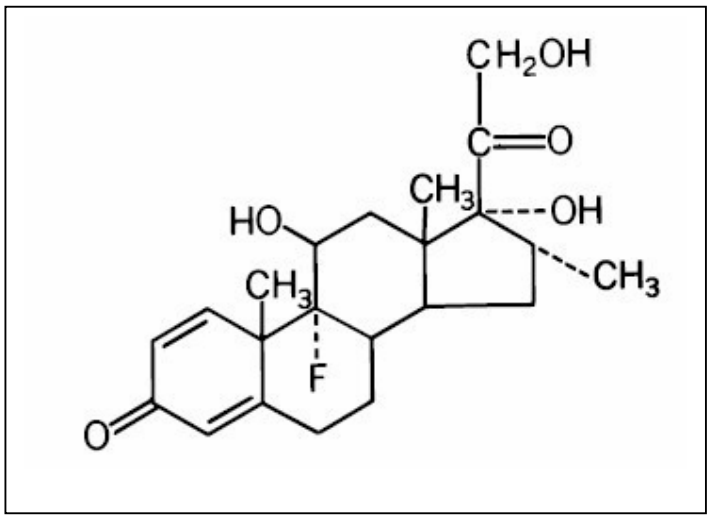
1. Allergic and hypersensitivity reactions

   Due to the preservative used – methyparaben

2. CVS : Bradycardia, hypotension

PHARMACOLOGY OF DEXAMETHASONE

Dexamethasone Sodium Phosphate Injection, is a synthetic adrenocortical steroid anti-inflammatory drug. It has got the following structure.



Dexamethasone Sodium Phosphate Injection, has a molecular weight of 516.41 and the chemical name 9-fluoro-11β,17,21-trihydroxy-16α-methylpregna-1,4-diene-3,20-dione 21-(dihydrogen phosphate) disodium salt.

Dexamethasone Sodium Phosphate Injection, 4 mg/mL is a sterile solution for intravenous, intramuscular, intra-articular, intralesional and soft tissue administration.

Mechanism of action:

The primary anti-inflammatory action is through inhibition of prostaglandin and leukotriene synthesis, increased antioxidant activity, and cell membrane stabilisation. It depresses formation, release, and activity of endogenous mediators of inflammation, including prostaglandins, kinins, histamine, liposomal enzymes, and complement expression. Also modifies the body's immune response.

Pharmacodynamics:

The half lives after intravenous and intramuscular administration were 5.4 hours and 7.4 hours, respectively. It is metabolized in the liver by the enzyme cytochrome P-450

AVAILABLE ROUTES:

Intravenous,intramuscular and intraarticular preparations are available.

DOSAGE:

Dexamethasone- all other inflammatory conditions:

Initial dose PO 0.75 to 9 mg/day.

Indications

Dexamethasone is indicated for short-term management of pain mainly, postoperative pain and musculoskeletal pain. It can be

used with opioid analgesic in general anaesthesia and as adjuvant for local anaesthetic in IVRA and apart from these they can be used in

a. Allergic states
b. dermatologic diseases.
c. endocrine disorders.
d. gastrointestinal diseases-ulcerative colitis.
e. hemotological diseases
f. neoplastic diseases.

Contraindications:

Depending on the indication and the general condition, peptic ulcers, osteoporosis, psychoses, infectious diseases, fungal infections diabetes and hypertension can be considered contraindications.

ADVERSE EFFECTS:

Some of the adverse effects seen with respect to individual systems.

Adverse Reactions

Fluid and electrolyte disturbances:

Sodium retention

Fluid retention

Congestive heart failure in susceptible patients

Potassium loss

Hypokalemic alkalosis

Hypertension

Musculoskeletal:

Muscle weakness

Steroid myopathy

Loss of muscle mass

Osteoporosis

Vertebral compression fractures

Aseptic necrosis of femoral and humeral heads

Pathologic fracture of long bones

Tendon rupture

Gastrointestinal:

Peptic ulcer with possible subsequent perforation and hemorrhage Perforation of the small and large bowel, particularly in patients with inflammatory bowel disease

Pancreatitis

Abdominal distention

Ulcerative esophagitis

Dermatologic:

Impaired wound healing

Thin fragile skin

Petechiae and ecchymoses

Erythema

Increased sweating

May suppress reactions to skin tests

Burning or tingling, especially in the perineal area (after I.V. injection)

Other cutaneous reactions, such as allergic dermatitis, urticaria, angioneurotic edema

Neurologic:

convulsions

Increased intracranial pressure with papilloedema (pseudotumor cerebri) usually after treatment

Vertigo

Headache

Psychic disturbances

Endocrine:

Menstrual irregularities

Development of cushingoid state

Suppression of growth in children

Secondary adrenocortical and pituitary unresponsiveness, particularly in times of stress, as in trauma, surgery, or illness

Decreased carbohydrate tolerance

Manifestations of latent diabetes mellitus

Increased requirements for insulin or oral hypoglycemic agents in diabetics

Hirsutism

Ophthalmic:

Posterior subcapsular cataracts

Increased intraocular pressure

Glaucoma

Exophthalmos

Metabolic:

Negative nitrogen balance due to protein catabolism

Cardiovascular:

Myocardial rupture following recent myocardial infarction

Other:

Anaphylactoid or hypersensitivity reactions

Thromboembolism

Weight gain

Increased appetite

Nausea

Malaise

Hiccups

Rare instances of blindness associated with intralesional therapy around the face and head

Hyperpigmentation or hypopigmentation

Subcutaneous and cutaneous atrophy

Sterile abscess

Postinjection flare (following intra-articular use)

Charcot-like arthropathy

Precautions

Renal Function

Use cautiously; monitor renal function.

Adrenal suppression

Prolonged therapy may lead to hypothalamic-pituitary-adrenal suppression.

Fluid and electrolyte balance

Can cause elevated BP, salt and water retention, and increased potassium and calcium excretion. Dietary salt restriction and potassium supplementation may be needed.

GI

Because of increased risk of perforation, use with caution in patients with diverticulitis, fresh intestinal anastomosis, latent peptic ulcers, or nonspecific ulcerative colitis.

Infections

May mask signs of infection. May decrease host-defense mechanisms to prevent dissemination of infection.

Stress

Increased dosage of rapidly acting corticosteroid may be needed before, during, and after stressful situations.

REVIEW OF LITERATURE

1. **ANAESTHESIA AND ANALGESIA 2006; 102:605-608**

Dexamethasone Improves the Quality of Intravenous Regional Anaesthesia and Analgesia. A Randomized, Controlled Clinical Study :

Zekiye Bigat, Neval Boztug, Necmiye Hadimioglu, Niha conducted a randomized controlled study and in this 75 patients were divided into three groups and received plain lignocaine-(group L,) (IVRA), lignocaine with dexamethasone in IVRA (group LD) and lignocaine with dexamethasone intravenously in the opposite arm. LDc The volume was 40 ml of 0.5 % lignocaine in each of the groups. Patients in group LD reported significantly lower pain scores and required less acetaminophen in the first 24 hrs after surgery. In Conclusion, the addition of 8 mg dexamethasone to lignocaine for IVRA in patients undergoing hand surgery improves postoperative analgesia during the postop period.

2. Anesthesia and Analgesia 1997;84:376–8."Epidural dexamethasone reduces the incidence of backache after lumbar epidural anaesthesia." YL.Yang,,, PP.Tang. SC,Tsai… CH Yang.

In this study 1000 patients were divided into 2 groups of Epidural lignocaine 2 %with dexamethasone and epidural

lignocaine 2 %. It was found that the patients receiving epidural lignocaine 2%. and dexamethasone had significantly lower pain scores

3. Anesthesiology 2002;96:1331–5

"The dose response and effects of Dexamethasone on bupivacaine microcapsules for intercostal blockade (T9–T11) in healthy volunteers.″Dan.J.Kopacz, Peter.G.Lacouter.Danlin Wu,Partha Nandy. In this study healthy volunteers were given Bupivacaine with Dexamethasone incorporated biodegradable microcapsules into the intercostal nerves. It was compared with plain Bupivacaine. The onset times, duration and efficacy of dexamethasone incorporated blocks were much higher when compared to the plain group.

4.ANAESTHESIA AND ANALGESIA 2002: 105; 481-486

"The prolonged post operative analgesia when Dexamethasone is added to a NSAID in Breast surgeries." Hval Kjetil, Thagaard K. Sem, Schlichting Ellen, and Raeder Johan, In this study Dexamethasone was found to prolong postoperative analgesia when added to an NSAID –Rofecoxib in a multimodal regimen.

. **5. ANAESTHESIA AND ANALGESIA: NOVEMBER 2002** .Intravenous Regional Anaesthesia using prilocaine and neostigmine: A. Turan, B. Karamanlyog M, D. Memis, G. Kaya and Z.

PaukcyDepartment of Anaesthesiology and Reanimation, Trakya University, Turkey. In this study, thirty patients undergoing hand surgery were randomly assigned to two groups to receive IVRA. They have used 0.5 mg of neostigmine as an additive. They found shortened sensory and motor block onset times, improved quality of anaesthesia and prolonged sensory and motor block recovery times, and prolonged time to first analgesic requirement in the neostigmine groups.

. **6. CANADIAN JOURNAL OF ANAESTHESIA SEPTEMBER 2006** : "Epidural dexamethasone reduces postoperative pain and analgesic requirements" **I**n this study 94 patients undergoing laparoscopic cholecystectomy were administered epidural blockade with group 1 given intravenously Dexamethasone and epidural Bupivacaine 0.25 % .Group 2 was given epidural Dexamethasone and Bupivacaine 0.25%. and group 3 was given epidural dexamethasone alone. It was concluded that groups receiving epidural dexamethasone had prolonged post operative analgesia and decreased opioid requirements when compared to the intravenous dexamethasone groups.

7. Anesthesia and Analgesia 2002;95:457-460

An evaluation of the analgesic efficacy of intravenous regional anaesthesia with lidocaine and ketorolac using a forearm versus upper arm tourniquet. Scott s. Reuben, MD., Robert B. Steinberg, MD, PhD, Holly Maciolek, RN and Poornachandran Manikantan MD, Department of Anaesthesiology, Tuffs University School of Medicine, Massachusetts. In this study, they assessed the analgesic efficacy of administering IVRA by administrating lidocaine and ketorolac with either a forearm or upper arm tourniquet for outpatient hand surgery. They concluded that forearm tourniquet intravenous regional anaesthesia with 0.5% lidocaine and ketorolac provides both a longer duration of sensory block and prolonged postoperative analgesia compared with upper arm IVRA. found in neostigmine group.

8. ANAESTHESIA AND ANALGESIA 2006 102: 263-267

Dexamethasone Added to Lignocaine Prolongs Axillary Brachial Plexus Blockade Sixty patients scheduled for elective hand and forearm surgery had axillary brachial plexus block were randomly allocated to receive either 34 mL lidocaine 1.5% with 2 mL of isotonic saline chloride (control group,) or 34 mL lidocaine 1.5% with 2 mL of Dexamethasone (8 mg) (dexamethasone group), Neither

epinephrine nor bicarbonate was added to the treatment mixture. The duration of surgery and the onset times of sensory and motor block were similar in the two groups. The duration of sensory (242 ± 76 versus 98 ± 33 min) and motor (310 ± 81 versus 130 ± 31 min) blockade were significantly longer in the dexamethasone than in the control group ($P < 0.01$). It was concluded that the addition of dexamethasone to lidocaine 1.5% solution in axillary brachial plexus block prolongs the duration of sensory and motor blockade.

9. ANESTHESIA AND ANALGESIA 2006; 88:430-44

0.5 % versus 1.0% 2 – chlorprocaine for Intravenous Regional Anaesthesia : A prospective randomized, Double – Blind Trial. Stephan C. Marsch, MD, D Phil, Mathias Sluga, MD, Wolfgang studier, MD, Jonas Barandun, MD, Domenic Scharplatz, MD and wolf gang Ummentioter MD, From the Departments of Anaesthesia and surgery, Switzerland. In this randomized prospective double – blind study they tested the hypothesis that compared 40 ml chloroprocaine 0.5 % with 40 ml chloroprocaine 1 % and found out that 1 % chloroprocaine results in an earlier onset of analgesia duration and improves distal tourniquet tolerance during IV regional anaesthesia. These beneficial effects must be weighed against a fourfold increase in signs of systemic local anaesthetic toxicity.

MATERIALS AND METHODS

This is a prospective double blind study conducted at Government Rajaji Hospital attached to Madurai Medical College.

After approval by the ethical committee 75 patients of ASA grade I & II of ages between 15-70 years who came for upper limb surgeries which lasted for less than 60 minutes were included in this study.

Patients with history of allergy to local anaesthetics, sickle cell disease, Raynaud's disease, scleroderma, local infection, Pagets disease and patients with inadequate starvation < 6 hours and patients who had contraindication to Dexamethasone were excluded from this study. Pre anaesthetic evaluation was done.

All patients were premedicated with Inj. Midazolam 2 mg IM 45 minutes before surgery. Resuscitation equipment and drugs were kept ready. Initial PR, BP, SPO2 were estimated continuously.

A 22 G cannula was placed intravenously as distal as possible in the arm to be anaesthetized. Venous access is established in the opposite arm to allow administration of fluids or drugs if necessary. The double tourniquet was applied on the arm with generous layers of padding, ensuring that no wrinkles are formed and the tourniquet edges do not touch the skin. The arm was exsanguinated by using

Esmarch bandage. If this was impossible, exsanguination was achieved by elevating the arm for 2-3 minutes.

The proximal tourniquet was inflated to at least 100 mm Hg higher than the patients systolic blood pressure. Before injecting local anaesthetic, radial pulse was palpated and confirmed that there was no pulse. The local anaesthetic is then injected slowly over 90 secs. A standard volume of 40 ml of 0.5% lignocaine or 40 ml of 0.5 mg lignocaine with dexamethasone was injected.

Patients were divided into three groups according to the drug which they received. Group A- (CONTROL GROUP) patients received 40 ml of 0.5% lignocaine, group B patients(SUDY GROUP) received 40 ml of 0.5% lignocaine with 8 mg of Dexamethasone and Group C(COMPARITIVE GROUP) patients received 40 ml of lignocaine with 8mg Dexamethasone intravenously in the opposite arm before the tourniquet was applied. After achieving surgical anaesthesia, the distal tourniquet which overlies part of the anaesthetized arm was inflated and the proximal one was deflated. After that the surgeons were allowed to proceed.

Intraoperatively PR, BP, SPO2, signs of drug toxicity were monitored regularly. If patient complained of tourniquet pain, they were supplemented with Inj. Midazolam IV (In titrated doses, max.

upto 2 mg) and intercostobrachial N block with local infiltration around the cuff. The cuff was not deflated until 20 minutes after local anaesthetic injection even if surgery was completed before 20 minutes. Cuff deflation was performed in cycles with deflation / inflation times of less than 10 seconds until the patient no longer exhibited signs of systemic toxicity. Patients were observed for 30 minutes after surgery.

Intraoperatively the following parameters were noted :

1.Onset of sensory and motor blockade.

2.PR, BP, SPO2 were monitored regularly at frequent intervals.

3.Duration of surgery

4.Need of supplementation for tourniquet pain

5.Side effects

6.Duration of blockade after cuff deflation both sensory & motor

Post operatively the following parameters were noted :

1.Time for first analgesic need.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002)

developed by Centers for Disease Control and Prevention (CDC), Atlanta for W.H.O.

Using this software, frequencies, percentage, range, mean, standard deviation, x^2 and 'p' values were calculated. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND OBSERVATIONS

A: Profile of cases studied

Table 1 : Age distribution

Age Distribution	Group A Control group		Group B Study group		Group C Comparative group	
	No.	%	No.	%	No.	%
Upto 20 yrs	1	4	4	16	7	28
21-30	11	44	10	40	8	32
31-40	11	44	7	28	5	20
>40	2	8	4	16	5	20
Total	25	100	25	100	25	100
Range	18-45 yrs		18-53 yrs		19-56 yrs	
Mean	30.9		30.7		30.1	
SD	5.8		9.7		11.0	
'P' value for A.B & C A & B A & C B& C	0.4517 Not Significant 0.4086 Not Significant 0.228 Not Significant 0.6265 Not Significant					

Comments:

The difference between the groups with respect to age is not statistically significant. Hence the groups are comparable with respect to age.

Table 2 : Sex Distribution

SEX	Group A Control group		Group B Study group		Group C Comparative group	
	No.	%	No.	%	No.	%
Male	16	64	12	48	17	68
Female	9	36	13	52	8	32
'p' value for						
A & B	0.3927 Not Significant					
A & C	0.7676 Not Significant					
B & C	0.2517 Not Significant					

The difference between the groups with respect to sex is not statistically significant. Hence the groups are comparable with respect to sex.

Table 3 : Weight (in Kgs)

Weight (in kgs)	Group A Control group	Group B Study group	Group C Comparative group
Range	42-60 yrs	40-59 yrs	40-60 yrs
Mean	52.2	50.5	49.8
SD	5.1	6.0	6.5
'P' value for			
A.B & C	0.4517 Not Significant		
A & B	0.4086 Not Significant		
A & C	0.228 Not Significant		
B& C	0.6265 Not Significant		

The difference between the groups with respect to weight is not statistically significant. Hence the groups are comparable with respect to weight

B: Efficiency of the three groups

Table 4: Onset of sensory Block

Onset of Sensory Block (in minutes)	Group A(mins) Control group	Group B(mins) Study group	Group C(mins) Comparative group
Range	2-9	2-7	2-7
Mean	4.41	4.52	4.82
SD	1.55	1.36	1.14
'P' value for			
A.B & C	0.3269 Not Significant		
A & B	0.7406 Not Significant		
A & C	0.1479 Not Significant		
B& C	0.2918 Not Significant		

The onset of sensory blockade in Study Group was 4.52 ± 2.72 and control group was 4.41 ± 3.10 mins in the control group and 4.82 ± 2.28 mins in the Comparative groups respectively. Though the effects of dexamethasone appears to be superior, the effect was not statistically significant. The effects were almost same.

Table: 5 - Onset of Motor Block (in minutes)

Onset of Motor Block (in minutes)	Group A (Control group) mins	Group B (Study group) mins	Group C (Comparative group)mins
Range	4-13	4-11	6-12
Mean	7.18	7.32	7.74
SD	2.51	1.68	1.36
'P' value for			
A.B & C	0.445 Not Significant		
A & B	0.5748 Not Significant		
A & C	0.1964 Not Significant		
B& C	0.5042 Not Significant		

The onset of motor blockade in study group was 7.32 ± 3.36mins and control group was 7.18 ± 3.02 and 7.74 ± 2.72 minutes respectively which was statistically not significant.

Table 6 : Duration of Surgery (in minutes)

Duration of Surgery (in minutes)	Group A (Control group)	Group B (Study group)	Group C (Comparative group)
Range	35-55	30-56	34-56
Mean	46.0	46.24	44.04
SD	5.76	6.16	6.85
'P' value for			
A.B & C	0.4754 Not Significant		
A & B	0.8374 Not Significant		
A & C	0.3208 Not Significant		
B& C	0.2752 Not Significant		

The duration of surgery in study groups were 46..24 ± 12.32 and 46.04±11.52 mins in the control group and it was 44.04 minutes in the comparative groups respectively. The difference between the groups with respect to duration of surgery is not statistically significant. Hence the groups were comparable with respect to duration of surgery.

Table 7 : Duration of sensory block after cuff deflation (in minute)

Duration of sensory block after cuff deflation (in minute)	Group A Control group	Group B Study group	Group C Comparative group
Range	3-7	3-14	2-10
Mean	4.94	7.8	5.12
SD	1.53	2.5	2.11
'P' value for			
A.B & C	0.0001 Significant		
A & B	0.0001 Significant		
A & C	0.7757 Not Significant		
B& C	0.0003 Significant		

The duration of sensory blockade after cuff deflation in study groups were 7.8±5.0 mins and 4.94 ±3.06 mins in the control groups and it was 5.12 ±4.24 mins in the comparative groups respectively.The duration of sensory blockade was longer in the study group and it was statistically significant,

Table 8 : Duration of motor block after cuff deflation

Duration of Motor block after cuff deflation (in minutes)	Group A (Control group)	Group B (Study group)	Group C (Comparative group)
Range	4-16	5-20	6-13
Mean	7.56	11.4	8.4
SD	2.52	3.39	2.0
'P' value for			
A.B & C	0.0001 Significant		
A & B	0.0001 Significant		
A & C	0.1008 Not Significant		
B& C	0.0006 Significant		

The duration of motor blockade after cuff deflation in study group was 11.4 ± 6.78 and 7.56 ± 5.04 in the control group and it was 8.4 ± 4.0 minutes in the comparative group respectively. The duration of blockade in the study group was found to be longer than that of control group and it was found to be statistically significant.

Table 9 : First Analgesic dose requirements

First Analgesic dose requirements	Group A Control group (mins)	Group B Study group (mins)	Group C Comparative group (mins)
Range	12-24	90-300	18-40
Mean	19.96	167.12	29.04
SD	9.26	48.54	6.12
'P' value for			
A.B & C	0.0001 Significant		
A & B	0.0001 Significant		
A & C	0.0001 Significant		
B& C	0.0001 Significant		

The time for first analgesic requirements in study group was 167.12 ± 97.08 and 29.04± 12.24 mins in the comparative group and 19.96 ± 18.52 mins in the control groups respectively. The differences between the study group and the control group was statistically significant. The lowest duration achieved in the study group was 90minutes and longest duration was 300 minutes.

Table 10 : Tourniquet pain supplementation

Tourniquet pain supplementation	Group A (Control group)		Group B (Study group)		Group C (Comparative group)	
	No.	%	No.	%	No.	%
Yes	15	60	5	20	13	52
No	10	40	20	80	12	48
'p' value for A & B A & C B & C	0.0094 Significant 0.7757 Not Significant 0.0392 Significant					

In the study group 20% patients (5patients out of 25 patients) needed supplementation due to tourniquet pain compared to 60%patients (15 patients out of 25patients) in the control group. The incidence of tourniquet pain in the study group was less and was statistically significant when compared to the control group.

Table 11: MAP changes after cuff deflation

MAP changes after cuff deflation	Group A Control group		Group B Study group		Group C Comparative group	
	Mean	SD	Mean	SD	Mean	SD
1 Minute	89.14	6.12	92.72	4.22	88.44	5.6
5 Minute	83.64	8.76	85.36	3.6	85.0	3.25
Change in MAP	8.46	5.32	7.36	5.24	5.6	4.47
% of Change	9.51	5.9	7.78	5.32	6.18	4.77
'P' value for						
A.B & C	0.1216 Not Significant					
A & B	0.4150 Not Significant					
A & C	0.0636 Not Significant					
B& C	0.214 Not Significant					

The difference between the study and the control groups with respect to mean arterial pressure at 1minute and at 5 mins is not statistically significant. Hence the groups were comparable with respect to mean arterial pressure at 1 minute and 5 mins after cuff deflation. There were no side effects noted in both the groups after cuff deflation.

Table 12 : Pulse Rate changes after cuff deflation

Pulse Rate changes after cuff deflation	Group A Control group		Group B Study group		Group C Comparative group	
	Mean	SD	Mean	SD	Mean	SD
1 Minute	76.42	13.65	81.96	6.9	87.28	6.32
5 Minute	73.96	4.82	83.64	4.23	78.84	5.55
Change in MAP	12.34	9.47	6.64	4.09	11.08	5.51
% of Change	15.89	12.06	8.28	5.52	12.5	5.92
'P' value for						
A.B & C	0.118 Not Significant					
A & B	0.1113 Not Significant					
A & C	0.497 Not Significant					
B& C	0.2232 not Significant					

The difference between the study and the control groups with respect to pulse rate which was recorded at 1 minute and 5minutes after cuff deflation was not statistically significant. Hence both the groups were comparable and the differences were not statistically significant. There were no side effects noted after cuff deflation in both the groups.

DISCUSSION

Intravenous regional anaesthesia uses local anaesthetics administered to one particular limb by occluding the arm proximally to provide conduction blockade. It must be safe, not threatening or unpleasant to the patient, allow adequate surgical access to the operative site, and cause as little disturbance as possible to the internal homeostatic mechanisms.

Local anaesthetic diffuses into the small veins surrounding the nerves and then into the vasa nervorum and capillary plexus of the nerves, leading to a core to mantle (centrifugal) conduction block in the nerves involved. Local anaesthetic then diffuses into the small nerves in the skin, blocking their conduction. The tourniquet produces ischaemia, which contributes to the analgesic action of the local anaesthetic by blocking nerve conduction and motor endplate function. 20 minutes after tourniquet application alone there will be analgesia to pinprick without the injection of any local anaesthetic. However, the speed of onset and the density of anaesthesia are greater with injection of local anaesthetic.

Intravenous regional anaesthesia has many advantages. It is simple, reliable with rapid onset and recovery. Despite these advantages intravenous regional anaesthesia has its own limitations

like lack of postoperative analgesia and tourniquet pain which causes discomfort to the patient. In this study, we attempted to eliminate these disadvantages by adding Dexamethasone as an adjuvant.

Comparison of results:

In this study, the three groups that is the control group (Group A) -lignocaine only, study group (Group B)- lignocaine with Dexamethasone and comparative group (Group C) lignocaine and intravenous-Dexamethasone in the opposite arm,in all these three groups patients were comparable with respect to age, sex, weight and duration of surgery.

Onset of sensory and motor blockade:

The onset of sensory and motor blockade is similar in the three groups as compared to the original Zekiye Bigat study.

Duration of sensory and motor block:

The duration of sensory and motor block recovery times are much longer in the (study group)-Dexamethasone IVRA group when compared to the Dexamethasone intravenous group and the control group in my study.This is in contrast to the original study by Zekiye et al study.

Tourniquet Pain:

The incidence of tourniquet pain was 20 % in Dexamethasone IVRA group 55 % in Dexamethasone intravenous group and 60% in the control groups.This is a similar finding to the original study.

Time to request of first analgesic:

The time to request of first analgesic dose was mean time interval-167 mins in the Dexamethasone IVRA group and in the comparative group and in the control groups it was 39 and 23 mins respectively.In the original study the time to request for the first analgesic was earlier in the IVRA Dexamethasone group but the total analgesic consumption for the first day was less in the IVRA Dexamethasone group.This result also correlates with the original study of Zekiye bagat et al.The p value was 0.001 which was clinically significant.

Surgical trauma results in release of intracellular contents from damaged and inflammatory cells. Nociceptor stimulation causes a neurogenic response with release of mediators such as substance P and neurokinin A. This results in an “inflammatory soup” containing histamine, serotonin, bradykinin and metabolites of the cyclooxygenase and lipooxygenase pathways.

Dexamethasone depresses formation, release, and activity of endogenous mediators of inflammation,including prostaglandins kinins, histamine, liposomal enzymes and complement expression.

The result is decreased afferent nociceptive signals arising from the site of surgery. Whether interfering with the synthesis of inflammatory mediators has a preemptive analgesic role in preventing sensitization of nociceptors remains controversial. The role of Dexamethasone in the management of postoperative pain is well established. Clinical studies have demonstrated an enhanced analgesic effect from Dexamethasone when concentrated at a peripheral site compared to the systemic administration of the same drug. This would suggest a predominantly peripheral site of action. .

It may be, that by concentrating the dose of Dexamethasone at the site of surgery, either as part of IVRA or wound infiltration, the resulting analgesic benefit is longer lasting than the same dose administered parenterally. Presumably there is a persistent drug level in the tissues, and this could result in reduced systemic side effects.

SUMMARY

The aim of this study is to prove the effectiveness of Dexamethasone as an adjunct to IVRA.Its effectiveness in relieving tourniquet pain and post operative analgesia was also studied.

The study was conducted in Rajaji Hospital Madurai, both elective and emergency surgeries were included in this study.

The patients were divided into three groups according to the drug which they received. Group A (CONTROL GROUP) patients received 40 ml of 0.5% lignocaine, Group B patients(STUDY GROUP) received 40 ml of 0.5% lignocaine with 8 mg of Dexamethasone and Group C(COMPARITIVE GROUP) patients received 40 ml of lignocaine with 8mg Dexamethasone intravenously in the opposite arm.

The parameters noted were :

1. Onset of sensory and motor blockade.
2. PR, BP, SPO2 were monitored regularly at frequent intervals.
3. Duration of surgery
4. Need for supplementation in case of tourniquet pain.
5. Side effects
6. Duration of blockade after cuff deflation both sensory & motor

Post operatively the following parameters were noted :

1. Time for first analgesic need.

After the entire study was conducted, it was found that adding Dexamethasone to lignocaine in IVRA yielded the following findings.

1. Prolonged post operative analgesia
2. Reduced tourniquet pain incidence.
3. Better quality of analgesia intraoperatively.

CONCLUSION

Dexamethasone 8 mg which was added to Lignocaine for Intravenous regional anesthesia provides

1. Less incidences of tourniquet pain
2. Increases the duration of post operative analgesia
3. No significant increase in side effects and there was no haemodynamic changes.

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PROFORMA

Name : Age / Sex :

IP No. : Weight :

Diagnosis : ASA Risk :

Surgery :

Premedication :

1. Onset of action : Sensory :

   Motor :

2. Side effects noted :

3. Duration of surgery :

Time	Pre	5	10	15	20	30	40	60	75	90	120
HR											
BP											

4. Supplementation :

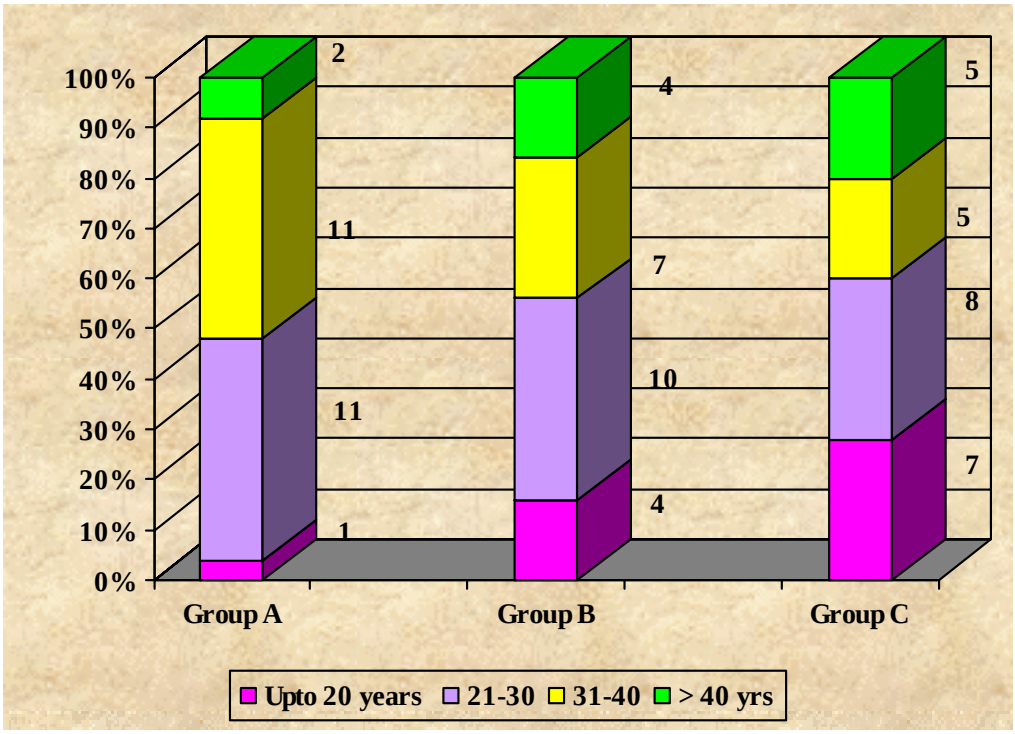
5. Duration of blockade after cuff deflation : Sensory :

   Motor :

6. Post operative :

   Time to first analgesic :

AGE DISTRIBUTION



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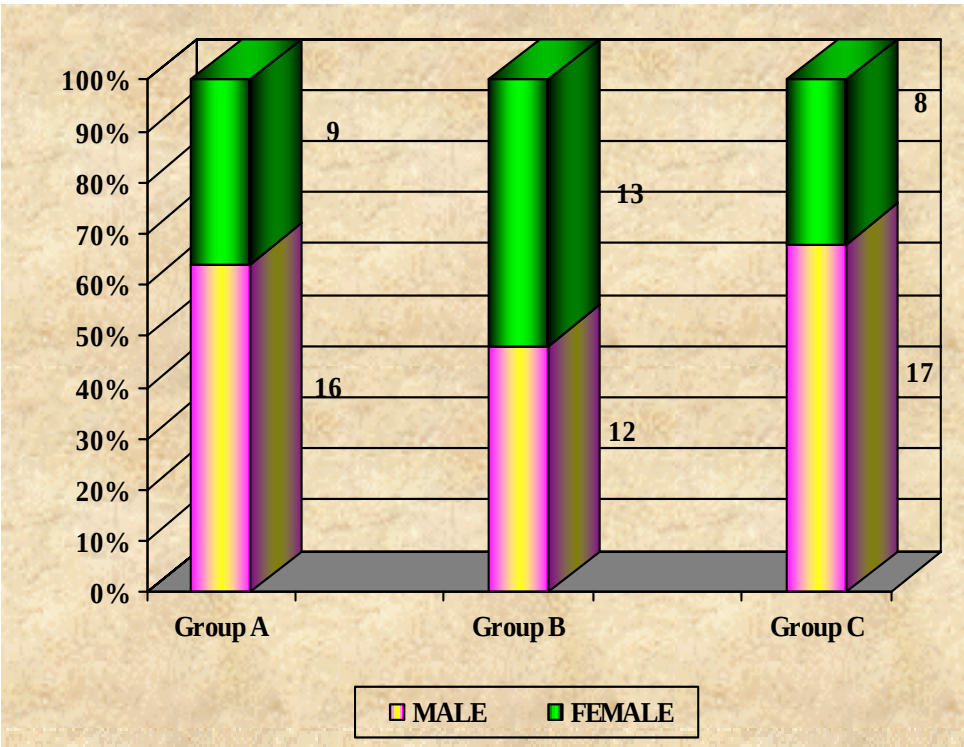

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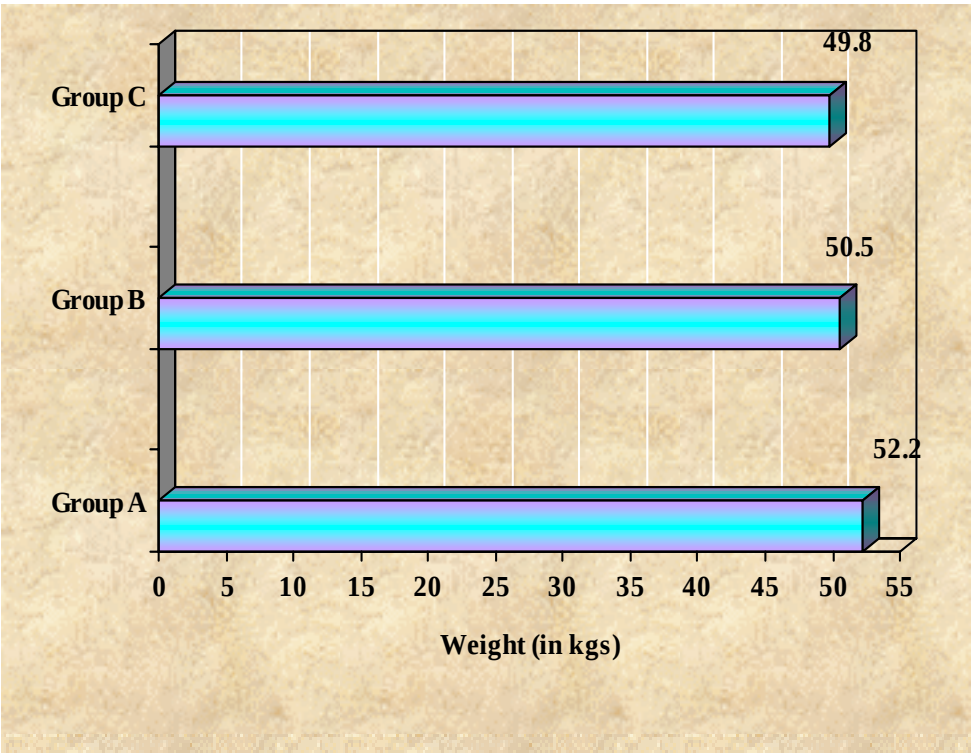
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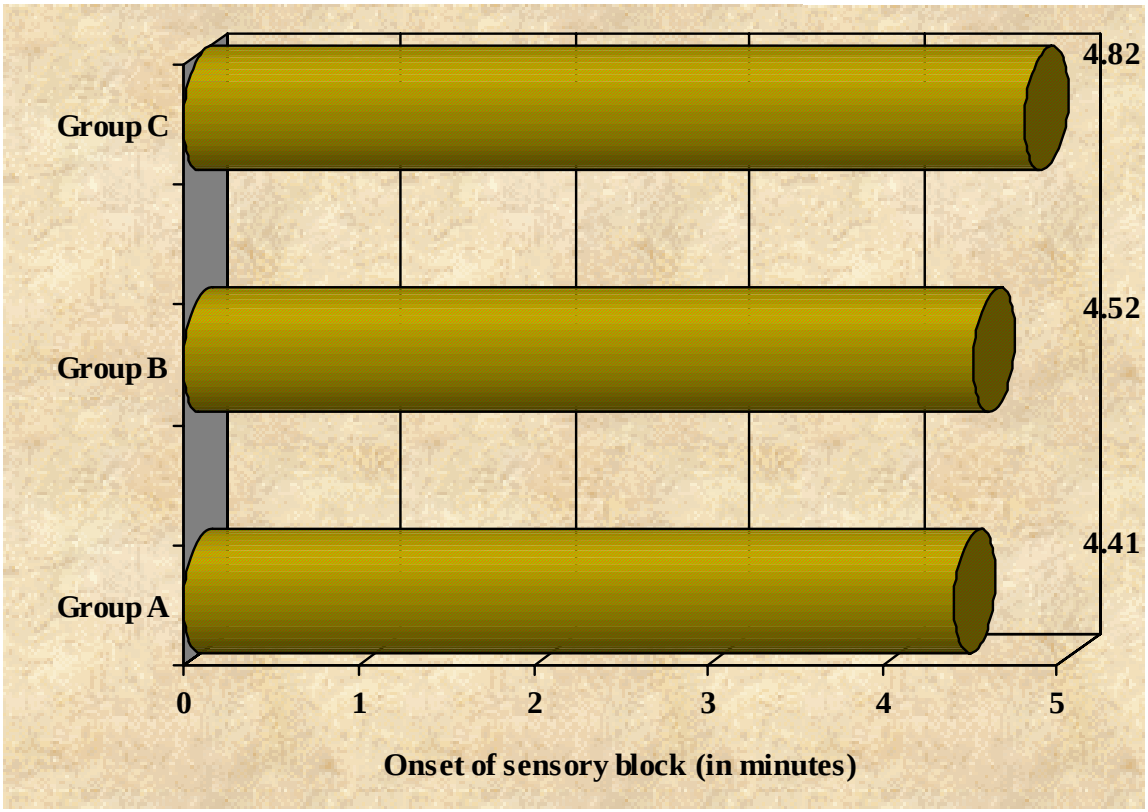
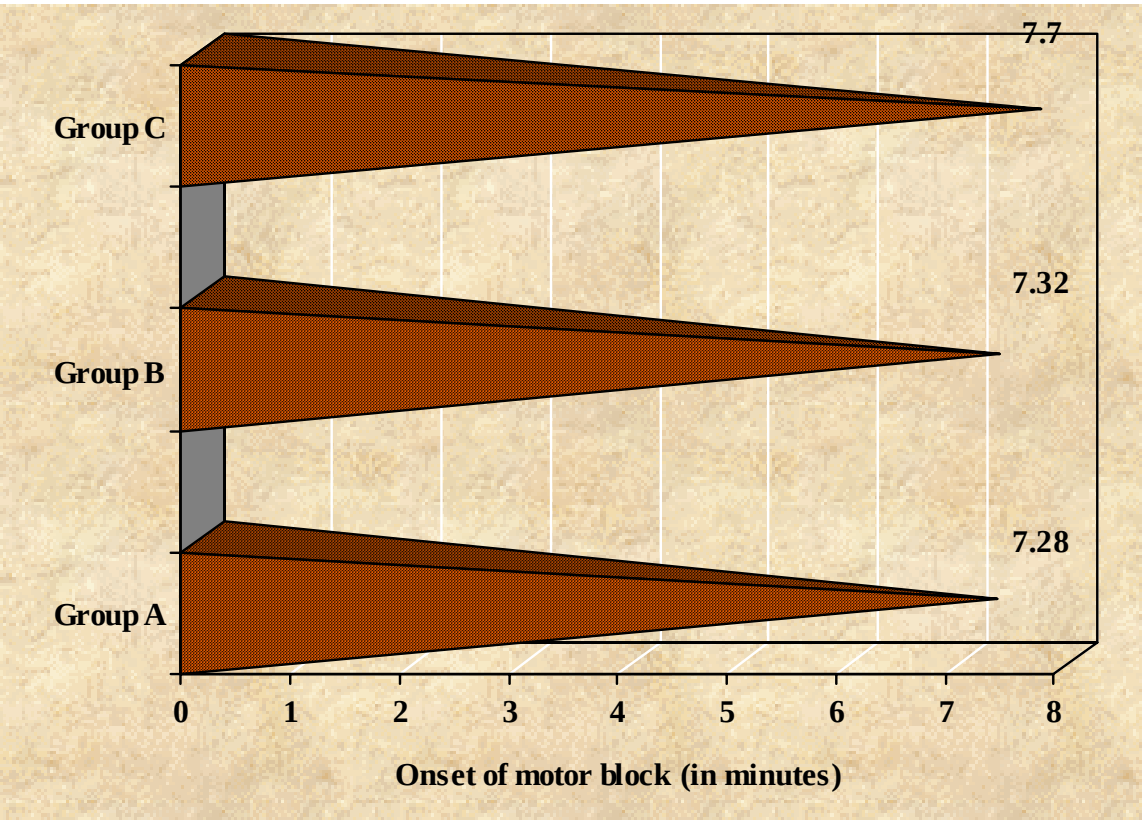
Group C
50.5
Group B
52.2
Group A
0
5
10
15
20
25
30
35
40
45
50
55
Weight (in kgs)

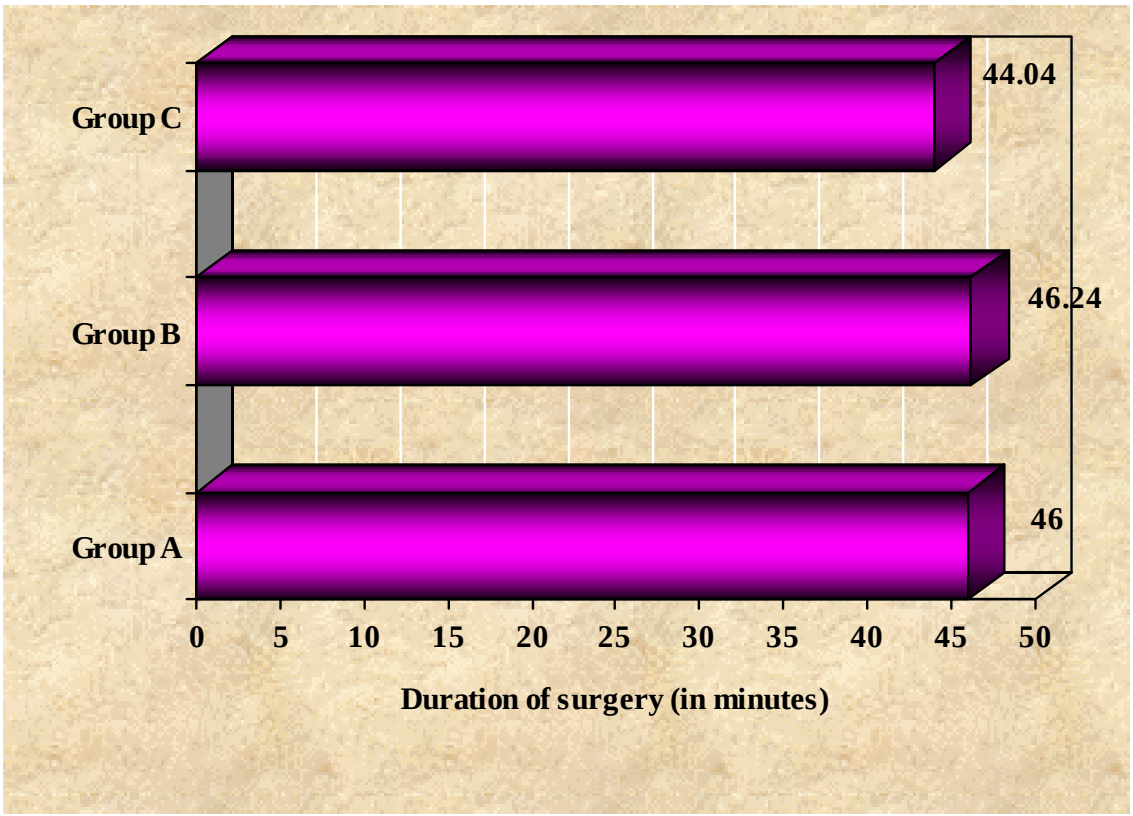
ONSET OF SENSORY BLOCK



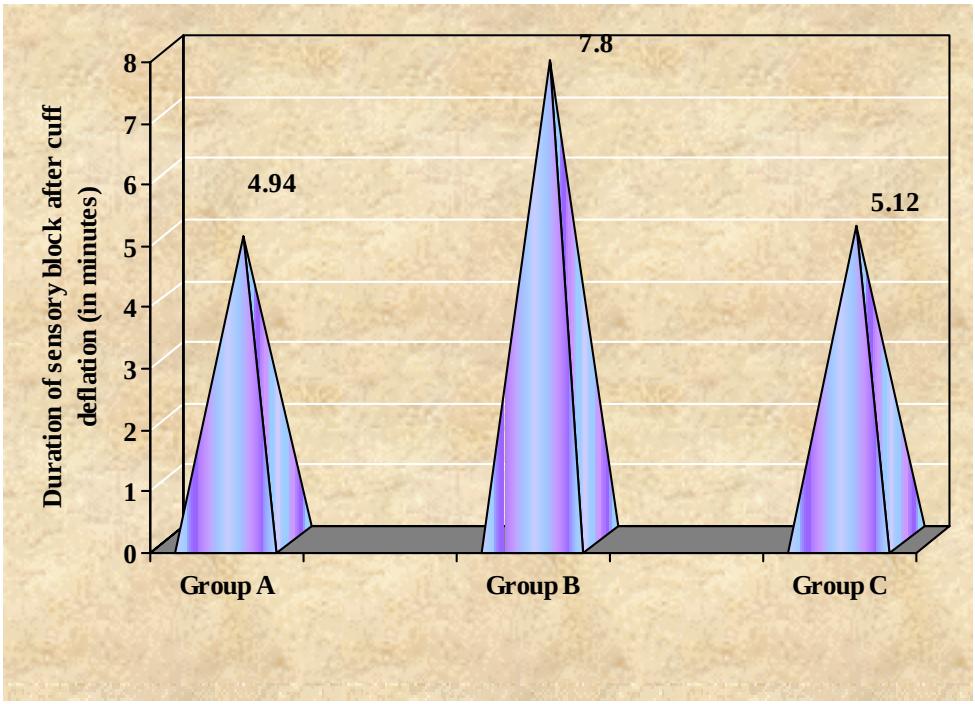
ONSET OF MOTOR BLOCK



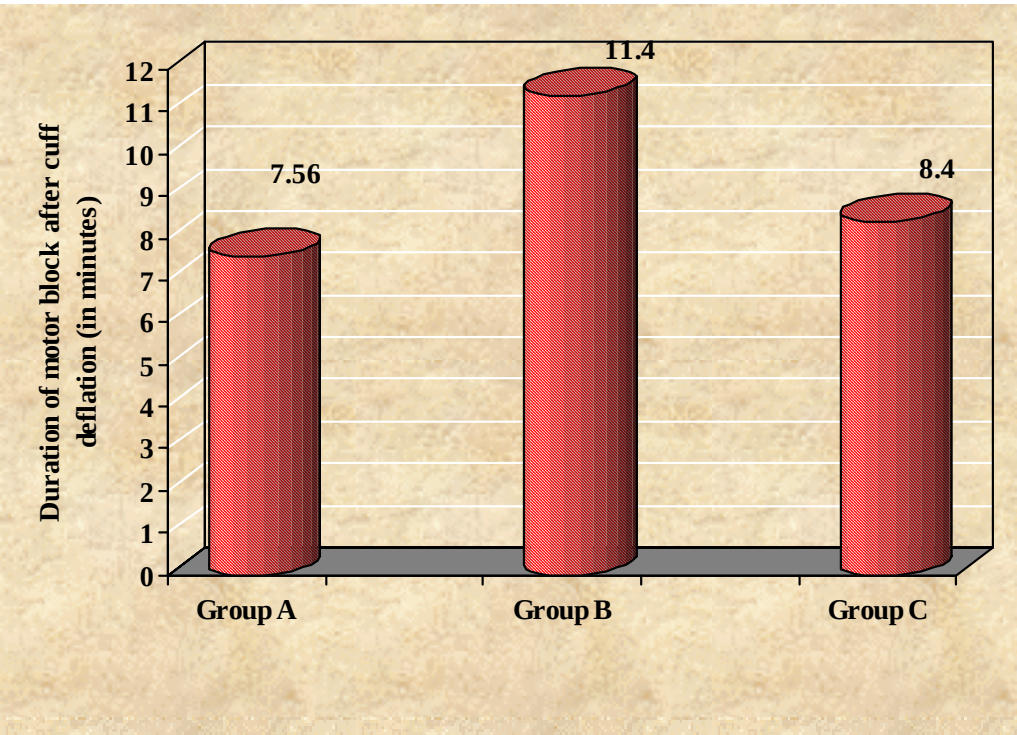
DURATION OF SURGERY



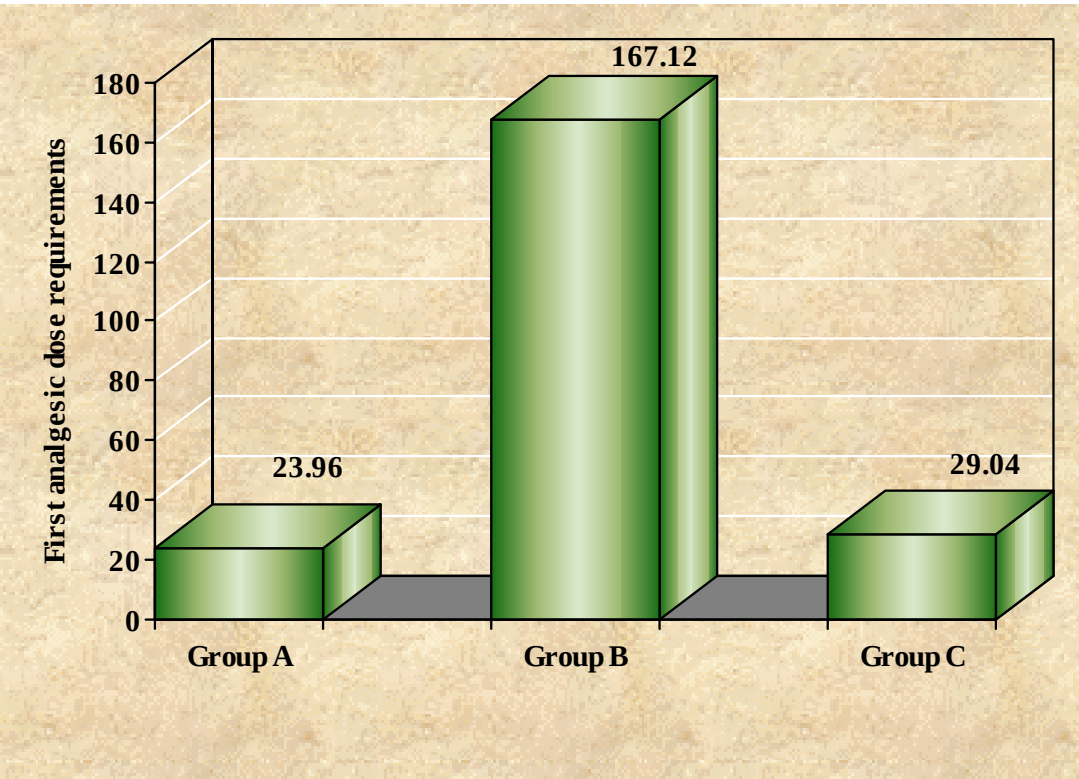
DURATION OF SENSORY BLOCK AFTER CUFF DEFLATION



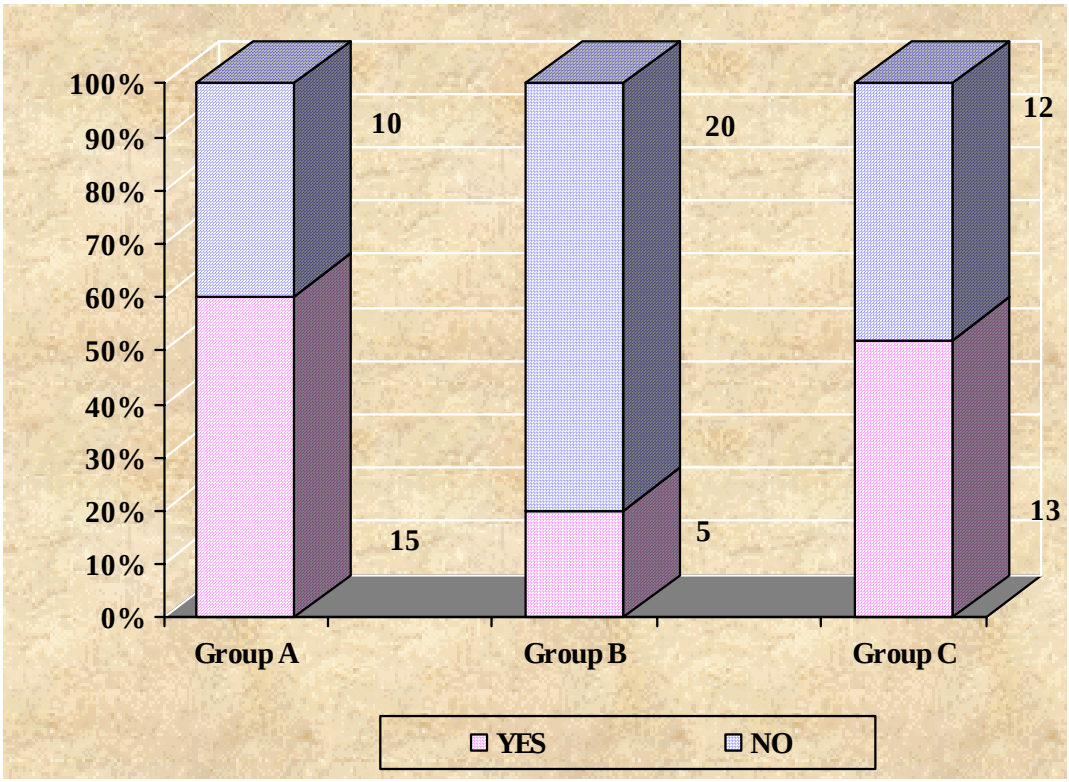
DURATION OF MOTOR BLOCK AFTER CUFF DEFLATION



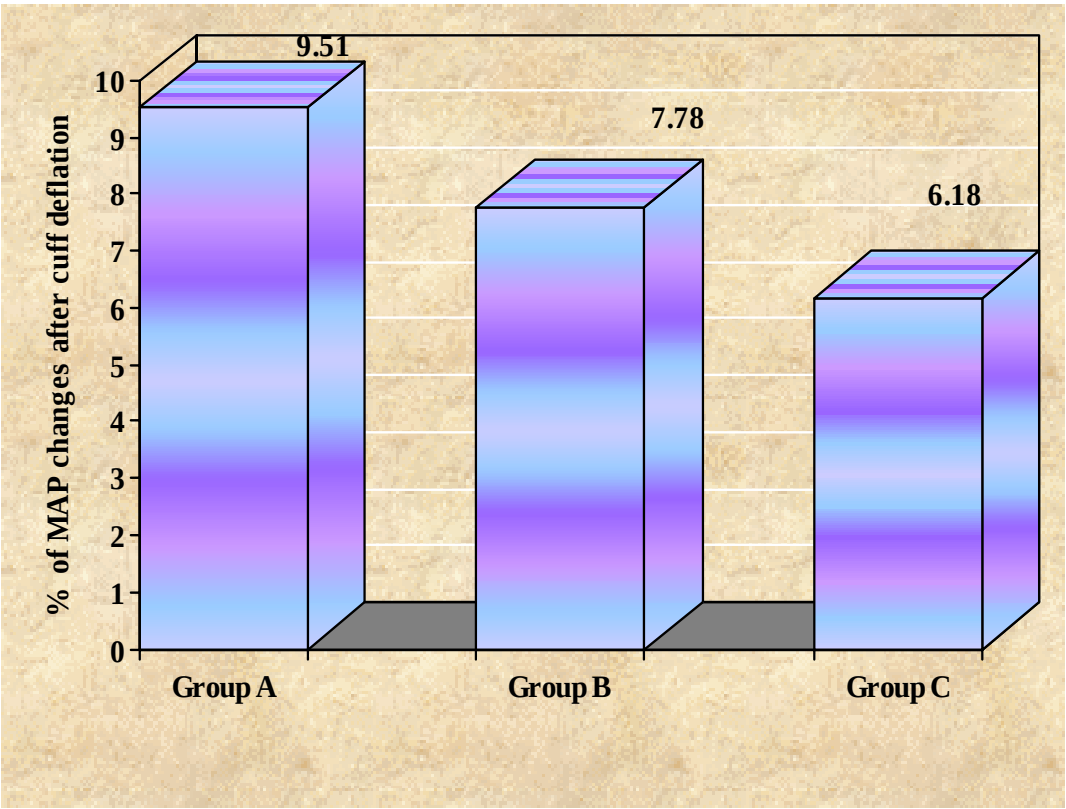
FIRST ANALGESIC DOSE REQUIREMENTS



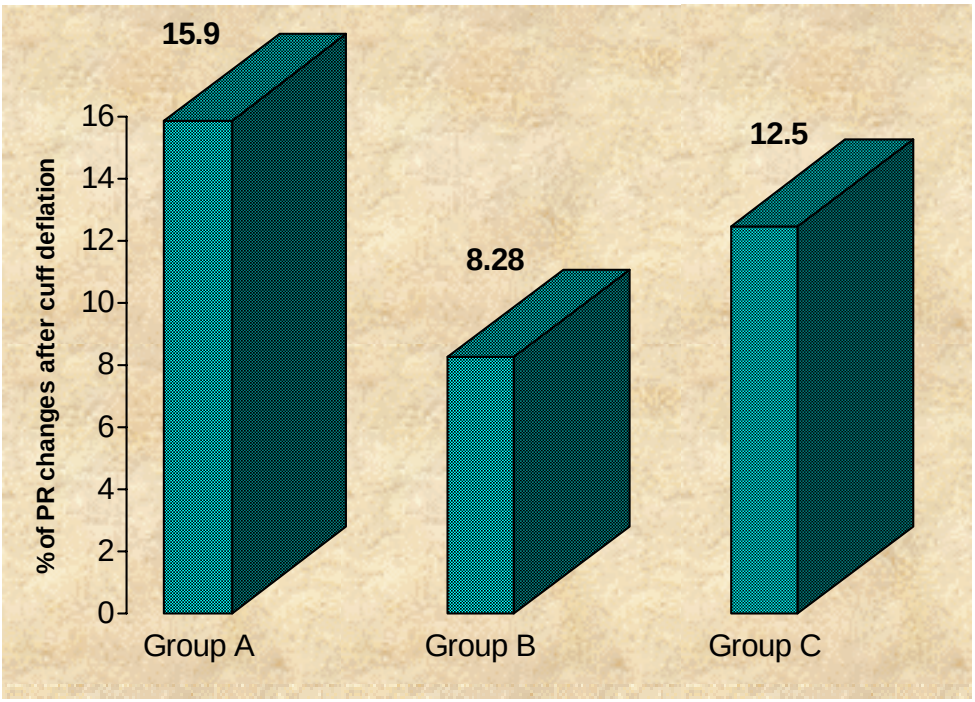
TOURNIQUET
PAIN SUPPLEMENT



% OF MAP CHANGES AFTER CUFF DEFLATION (in minutes)



% OF PULSE RATE CHANGES AFTER CUFF DEFLATION (in minutes)



S.NO		groups	SEX	AGE	WEIGHT	surgery	ASA	onset sensory block	onset motor block	duration of surgery	duration of sensory block after cuff deflation	duration ofmotorblockafter cuff deflation	requirement for first analgesic dose	Tourniquet pain supple mentation	MAP after cuff deflation at 1 min	MAP after cuff deflation at5min	pulse rate after cuff deflation at 1 min	pulse rate after cuff deflation at 5 mins
1	marimuthu	A	M	22	48	FTI	I	4	6	45	3	5	15	yes	82	71.5	92	70
2	nagammal	A	F	28	50	Gang	II	4	7	50	3.5	4.5	13	yes	82.5	72	61	92
3	chinna maruthu	A	M	34	52	Gang	I	2	9	40	4	5	12	no	90	71.5	70	72
4	pandi	A	M	33	56	Gang	I	5	5	35	3.5	5.5	12	no	91	71	62	74
5	rakku	A	F	35	58	Gang	I	4	5	48	4	5.5	14	yes	80	76	59	75
6	lakshmi	A	F	37	52	R Radius	II	4.3	5	50	4.5	6	13	yes	82	87	90	76
7	ponni	A	F	18	42	Gang	I	4	6	55	6	9	20	no	83	90	63.5	7
8	saroja	A	F	24	44	Gang	I	5	6	46	3	4	15	yes	83	96	64	72
9	kannan	A	M	29	54	Gang	I	6	13	39	7	9	14	no	83	90	62	70
10	gopal	A	M	41	59	Ext.Poi. L	II	7	9	43	6	9	20	yes	87	76	68	68
11	mageswari	A	F	45	45	Gang	I	9	11	47	7	9	23	no	88	84	70	70
12	kalaiyan	A	M	27	48	Gang	I	4	5	50	7	9	24	yes	98	85	94	72
13	thangaiah	A	M	34	60	RA FA	I	5	6	42	4	8	14	no	91.5	89	80	74
14	muthuveeran	A	M	26	52	Gang	I	6	8	44	7	10	20	yes	90	78	83	76
15	perumalaiya	A	M	29	54	ETI	I	2	4	50	7	10	23	no	84	79	60	80
16	pothum ponnu	A	F	32	56	FTI	I	4	6	53	4	7	22	yes	100	102	74	70
17	azhagar	A	M	34	54	Gang	I	3	5	38	3.5	7	19	no	102.5	103.5	94	72
18	natarajan	A	M	33	58	LFA	I	4	8	40	4	6.5	22	yes	96	84	100	74

19	senthil	A	M	28	59	C F3-F5	I	5	8	53	4	6	24	no	92	88	74	76
20	chinna ponnu	A	F	32	59	Gang	I	6	12	48	3.5	7	20	no	84	86	104	72
21	thenappan	A	M	32	48	Gang	I	4	6	52	4	7	21	yes	88	87	74	70
22	muthuraja	A	M	30	46	E Rt Ra	I	3	4	38	7	10	15	yes	92	79	70	80
23	ayeesha beevi	A	F	25	50	F Radi	I	3	9	40	4	6	21	yes	92.5	80	80	72
24	manimaran	A	M	35	52	Gang	I	4	8	52	6	8	17	yes	92.5	81.5	92	74
25	abdul saleem	A	M	30	50	Gang	I	3	11	52	7	16	166	yes	94	84	70	76
26	thangapandi	B	M	32	52	X Rt FA	I	3	5	46	7	14	160	yes	88	88	90	80
27	ravikumar	B	M	36	49	Gang	I	3	5	50	7	13	140	yes	87	82	85	82
28	pandiyammal	B	F	20	40	Gang	I	2	4	51	5	10	152	no	90	84	80	78
29	fathima beevi	B	F	18	40	Gang	I	4	7	56	6	12	165	no	92	0	82	83
30	balamurugan	B	M	20	42	Lip elbo	I	4	7	30	8	9	200	no	94	82	78	84
31	piroja	B	F	22	44	F BB F	I	4	8	35	9	12	176	no	83	82.5	76	82
32	kanimozhi	B	F	30	51	Neu Fib	I	3	6	50	11	18	110	no	96	83.5	74	82
33	jerina	B	F	32	54	F BB FO	II	4	6	52	4	5	90	no	97	88	72	82
34	thayaleeswaran	B	M	27	58	Gang	I	5	8	47	6	7	120	no	98	85	86	84
35	nagarajan	B	M	32	57	F T I	II	6	9	44	7	11	125	yes	94	79	80	88
36	rani	B	F	30	50	Gang	I	7	10	50	11	15	300	no	94.5	92	82	80
37	bakkiyam	B	F	22	43	Gang	I	5	11	52	9	12	195	no	94	91	90	82
38	saraswathi	B	F	24	49	Gang	I	5	7	40	4	12	220	no	93	90	82	80
39	selvi	B	F	30	51	RA FA	I	4	8	42	9	12	185	no	94	82	80	74

40	ponnammal	B	F	31	52	Gang	I	3	6	50	8	10	155	yes	102.5	84	86	80
41	rajeshwari	B	F	19	44	ETI	I	4	6	38	3	6	145	no	100	86	89	84
42	sasikumar	B	M	22	48	FTI	I	5	8	40	7.5	9	140	no	92	85	72	88
43	duraipandy	B	M	31	55	Gang	I	4	6	42	8	10	200	no	93	90	74	82
44	suseela	B	F	27	58	LFA	I	4	6	48	8	11	144	no	95	82	82	86
45	perumal	B	M	28	52	C F3-F5	I	4	8	50	7.5	10	210	no	90	84	84	88
46	baskar	B	M	40	52	Gang	I	4	7	52	8	11	90	no	90	84	76	90
47	murugesan	B	M	43	59	Gang	I	6	9	48	8	10	220	yes	88	82	77	92
48	rajamahendran	B	M	48	59	E Rt Ra	I	6	8	50	12	16	146	no	90	89	78	82
49	silambarasan	B	M	50	58	F Radi	I	7	9	43	8	10	150	no	92	81	96	88
50	muthumari	B	F	53	45	Gang	II	7	9	50	14	20	240	no	91	88	98	90
51	paulraj	C	M	40	52	Gang	I	4	7	52	3	7	28	no	81	86	90	81
52	chithra	C	F	42	50	X Rt FA	II	4.5	7	56	2	8	30	no	83	84	78	84
53	selvam	C	M	32	50	Gang	I	5	7	40	3	6	26	no	84	88	88	70
54	panneerselvam	C	M	20	40	ETI	I	5	7	42	2	7	31	yes	90	90	80	72
55	mohammed gha	C	M	21	42	FTI	I	5	7	37	4	9	30	yes	91	82	82	74
56	chelliah	C	M	20	50	Gang	I	4.5	6	34	2	7	32	yes	92	81	84	80
57	rose mary	C	F	32	52	LFA	I	6	10	53	3	7	25	yes	96	82	72	84
58	velliammal	C	F	30	41	C F3-F5	I	6	8	48	5	7	32	yes	98	84	80	84
59	krishnasamy	C	M	28	49	Gang	I	6	8	40	6	7	30	yes	93	88	82	84
60	rani	C	F	32	52	Gang	I	6	9	52	7	10	21	no	80	90	84	86

61	john peter	C	M	26	56	E Rt Ra	I	7	7.5	54	4	7	34	yes	82	84	83	88
62	muniammal	C	F	30	59	F Radi	I	4.5	7	39	5	11	28	no	90	82	88	70
63	mani	C	M	31	60	Gang	II	3	7	52	8	13	32	yes	91	90	86	72
64	selvam	C	M	20	42	Gang	I	3	7	54	10	12	40	no	92	82	89	74
65	pazhani	C	M	22	56	X Rt FA	I	3	7	39	4	6	26	no	94	84	90	76
66	ranjith mohan	C	M	46	45	F BB F	I	4	8	36	5	8	30	no	96	86	92	78
67	raman	C	M	48	49	Neu Fib	I	5	6	41	6	9	34	no	94	91	94	80
68	manimegalai	C	F	20	54	F BB FO	I	4	8	40	7	10	38	no	82	82	97	82
69	bashira	C	F	20	58	Gang	II	5	9	43	8	11	40	no	82	83	98	80
70	abdul kasim	C	M	19	41	F T I	I	6	10	50	5	7	31	no	90	84	96	82
71	devi	C	F	19	40	Gang	I	7	7	37	6	11	20	yes	82	82	93	80
72	raghunath	C	M	21	43	Gang	I	4	7	36	8	9	31	yes	84	82	88	72
73	sankaraiah	C	M	28	51	Gang	I	4	7	40	4	6	20	yes	82	86	90	74
74	subramani	C	M	50	56	RA FA	I	4	12	42	5	7	18	yes	92	90	92	76
75	rajan	C	M	56	58	Gang	I	5	7	44	6	8	19	yes	90	82	86	88